FAILED BACK SYNDROME

ETIOLOGY, CLINICAL PRESENTATION, DIAGNOSIS AND DIFFERENT METHODS OF TREATMENT

Thesis

Submitted In Partial Fulfillment For The Degree of M.D.
In Neurosurgery

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1999

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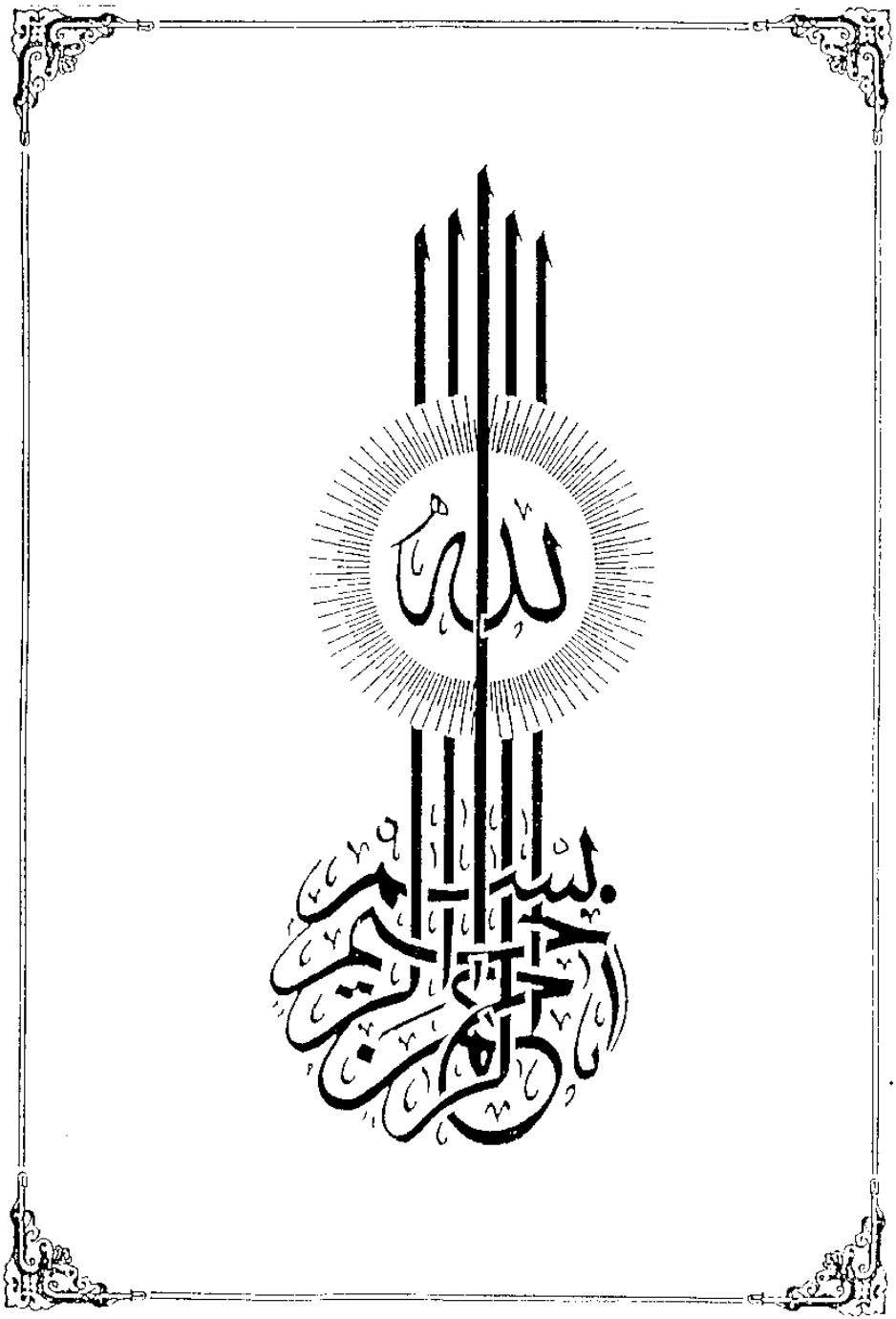

ACKNOWLEDGEMENT

In the start I would like to thank ***Prof. Dr. Hassanin El-Sherif,*** the Head of Neurosurgical Department in Faculty of Medicine. Ain Shams University, he had chosen this subject and greatly influenced every detail by this enthusiastic guidance, innovation and constructive criticism.

I would like to express my deepest gratitude to ***Prof. Dr. Mohamed Tawfik Hosny,*** Head of the Neurosurgical Department in Maadi Armed Forces Hospital. His kind supervision and continuous encouragement and help during the progress of this work.

My special thanks also for ***Prof. Dr. Hossam Mohamed El-Hussieny,*** who by his continuous encourgement, made the accomplishment of this work possible and contributed to its final shape.

Lastly, My great thanks to every member of neurosurgical department in Maadi-Armed Forces Hospital and Ani-Shams University Hospital, both seniors, and juniors, for their great help and support; without which this thesis would be impossible.

CONTENTS

Aim of the Work

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1- To define incidence of failed back syndrome

2- Searching for the etiology and clinical presentation of failed back syndrome.

3- To determine the recent methods of diagnosis.

4- To determine the methods of treatment of failed back syndrome.

Introduction

INTRODUCTION

"Failed back surgery syndrome" (FBSS), or its euphemism "Failed back syndrome", is persistent or recurrent low back pain, with or without sciatica, following one or more lumbar spine operations [*North, 1985*]. It reflects failure of lumbar spine surgery to relieve pain and incapacitation [*Burton, 1983d*]. Surgical procedures most commonly employed in the management of low back pain and/or sciatica are lumbar discectomy, decompressive laminectomy and fusion. Failure was frequently regarded by surgeons as due to specific complications; or as failure to achieve specific surgical objectives; for example, to reverse neurological deficits. However, when operating primarily for pain as in most low back surgery, "failure" to the patient usually means failure to improve pain or even causing its aggrevation.

Since the introduction of practical techniques of spine fusion by Albee and Hibbs in 1911 and the concept of the prolapsed intervertebral disc by *Mixter and Barr in 1934*. Innumerable patients have had spinal operations for low back pain and sciatica. It is inevitable, of course, that these procedures are not always successful.

It is estimated that 65% of people will have one or more significant attacks of low back pain in their life time. These attacks occur primarily betwen the ages of 25 and 50 [*Livermore, 1984*]. Fortunately

95% of these attacks resolve spontaneously and the incidence of operation in low back pain problems is estimated to be less than 4% [*White, 1984*].

Low back pain might be second only to upper respiatory tract infection among the most common disease entities affecting the populace of developed nations today [*Livermore, 1984*]. Perhaps decreasing physical activity and increasing life span have made man more vulnerable to decompensation of low-back function [*Weinstein, 1982*].

Surgical failure rates for low back pain and/or sciatica have been reported as varying between 10 and 40 percent [*Burton, 1985*]. The high failure rates are usually attributed to residual backache rather than sciatica (Table 1). Also, surgery for low back pain alone has been less successful [*Spangfort, 1972 and Nelson, 1977*]. Clearly, such an outcome is of great concern in a disease that is benign, theortically correctable and tends to occur in the most productive segment of the society. This unfavourale outcome explains why patients with lumbar spine problems are often given dire warnings about surgical operations, not only from non-medical sources but even from some specialist surgeons [*Taylor and Akeson, 1971*].

Failed back surgery syndrome (FBSS) is one of the most difficult therapeutic challenges to medicine because it represents a complex interplay of organic, psychologic and socioeconomic factors. Invariably, the patient has been misdiagnosed and improperly treated, and the results are long term disability, drug dependancy, lack of productivity, behavioral

disturbance, and for some, social and economic catastrophe. FBSS is not only a failure of medical care but also represents, in terms of medical care costs and lost worker productivity, a huge financial liability for the patient as well as society in general.

The evaluation and treatment of patients with FBSS are becoming increasing problems. The sequlae of surgery are superimposed on whatever remains of the underlying problem. Compounding the difficultities of diagnosis. Such failures are usually compounded by emotional breakdown of the patient, either antedating or following surgery, further complicating the problem. The inherent complexity of these patients demands a method of problem solving that is precise and unambiguous. In order for the problem of FBSS to be sucessfully overcome, a multidisciplinary approach should be adopted, in which various specialities such as neurosurgery, orthopedic surgery, radiology, neurology, psychiatry, rheumatology, physiotherapy, and social work have to be included. This essay hopefully aims at covering the neurosurgical aspects of such a multidisciplinary approach.

Review of Literature

EMBRYOLOGY OF LUMBAR SPINE

The nervous system, with its associated structural support system, is clearly the most complicated anatomic system of the human body. It's 100 billion neurons and 1 trillion glial cells form a complex network of connections that constitute the structural frame-work of nature is most complicated piece of biologic architecture of the human body [*Last, 1970*].

Fetal development may be conceptually divided into three stages: pre-embryonic, embryonic and fetal. The pre-embryonic period begins at fertilization and lasts for approximately 3 weeks. The three primary germ layers (ectoderm, mesoderm, and endoderm) develop during this peroid. [*Crelin, 1976 and Bog duk, 1987*]. The second stage, embryonic period, is the most active period and the one in which the organ system form. It lasts from approximately the third to eighth week of development. The fetal period is the final stage of development and lasts from approximately eight weeks to birth. The organ systems formed during the embryonic period undergo further growth and development during this stage.

At approximately 16 days, the embryo is transformed from a two layered structure, consisting of a dorsally situated ectoderm and amniotic cavity and a ventrally situated endoderm and yolk sac, to a trilaminar structure. At this stage, a proliferation of mid line ectodermal cells becomes the primitive pit.

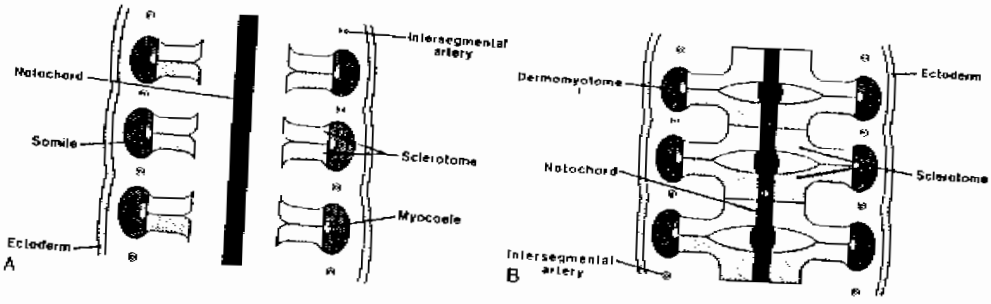

Fig. (1): Formation of vertebral column and related neuromuscular structures. A-C. Note that centrum is formed from equal contributions of two adjacent sclerotomes.

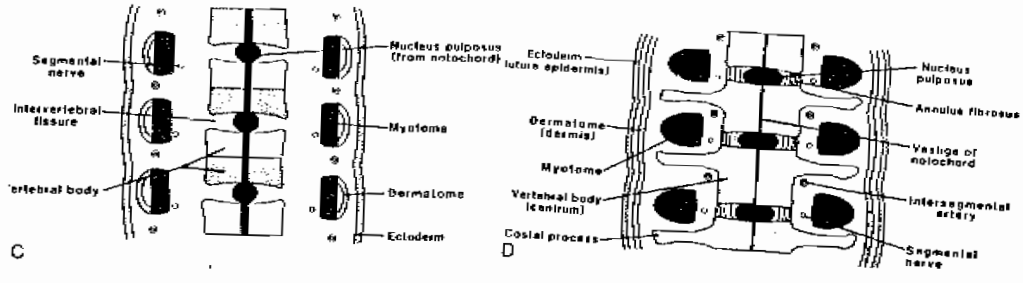

Fig. (2): Continued C,D the segmental artery, originally lying between sclerotomes, comes to lie opposite the centrum. D, the spinal nerve comes to lie opposite the intervertebral disc. Note that the intervertebral disc develops from notochordal tissue between centra, whereas notochordal tissue enclosed by the centrum ultimately degenerates (D).

The ectodermal cells adjacent to this opening are known as the primitive knot, and their cranial proliferation produces Hensens node [*Crelin et al., 1981*]. This tubular notochordal process is destined to become the notochord. The hollow cavity and ventral surface of the notochordal process disappear so that the dorsal surface of the process comes to lie in direct contact with the underlying endoderm and yolk sac, forming a flat notochordal plate. Further, in a craniocaudal direction, the lateral edges of the notochordal plate curl, nuiting the edges of the endoderm and producing the true notochord, which thus comes to lie between the underlying endoderm and overlying ectoderm [*Crelin, 1981*].

During the third week of fetal development, the ectoderm overlying the notochord thickens , producing the neural plate. The lateral edges of this plate proliferate dorsally and eventually curl over the neural plate to create a tubular structure known as the neural tube [*Lonstein, 1983*].

By the end of the third week, the third layer of the trilaminar embryonic structure forms. This layer, known as the mesoderm, is situated between the dorsal ectoderm and the ventral endoderm and lies lateral to the notochord. By approximately the 19th day, the mesoderm has divided into three distrinct areas by virtue of its complex differentiation in both a mediolateral and a craniocaudal direction. The most cellular portion of this mesodermal structure lies immediately lateral to the notochord and is known as the paraxial mesoderm [*Mac Nab, 1977*].